Patent Claims

1. A method for the prophylactic and/or therapeutic treatment of a receptor tyrosine kinase (RTK)-hyperfunction-induced disorder in a mammal, which method comprises administering to a mammal in need thereof an effective amount of at least one inhibitor of fibroblast growth factor receptor-4 (FGFR-4), wherein said RTK-hyperfunction-induced disorder is treated prophylactically and/or therapeutically.

2. The method of claim 1, wherein said RTK-hyperfunction-induced disorder is one or more disorders selected from the group consisting of cancer, a disease attributable to cellular hyperproliferation and/or cellular invasion of tissue, a carcinoma, and a metastasis.

3. The method of claim 2, wherein said disorder is breast cancer, squamous cell carcinoma, glioblastoma, neuroblastoma or uterine cancer.

4. The method of claim 1, wherein said inhibitor is a kinase-inactive receptor.

5. The method of claim 1, wherein an overexpression and/or an altered activity of FGFR-4 is lowered and/or inhibited.

6. The method of claim 5, wherein the overexpression and/or the altered activity of FGFR-4 is triggered by a mutation of the FGFR-4.

7. The method of claim 6, wherein the mutation is one or several point mutations.

8. The method of claim 7, wherein the one or several point mutations leads to an exchange of a hydrophobic amino acid for a hydrophilic amino acid.

9. The method of claim 6, wherein the mutation occurs in the transmembrane domain of FGFR-4.

10. The method of claim 9, wherein the mutation is one or several point mutations that lead to an exchange of a hydrophobic amino acid for a hydrophilic amino acid.

11. The method of claim 6, wherein the mutation occurs at amino acid position 388 in the FGFR-4 molecule.

12. The method of claim 11, wherein the mutation leads to an exchange of glycine for arginine.

13. The method of claim 6, wherein the mutation is a germ line mutation.

14. The method of claim 1, wherein the FGFR-4 is mutated and the inhibitor inhibits the mutated FGFR-4.

15. A method of diagnosing an RTK-hyperfunction-induced disorder or a genetic predisposition therefor in a mammal, which method comprises determining the presence of a mutated FGFR-4 protein or a nucleic acid encoding a mutated FGFR-4 protein in a sample of protein or nucleic acid, respectively, obtained from said mammal, wherein the presence of such a protein or nucleic acid is indicative of an RTK-hyperfunction-induced disorder or a genetic predisposition therefor.

16. The method of claim 15, wherein said RTK-hyperfunction-induced disorder is cancer.

17. The method of claim 15, which method comprises contacting the sample of nucleic acid with a labeled DNA or RNA molecule encoding a mutated FGFR-4 under hybridizing conditions and detecting the labeled DNA or RNA molecule after hybridization, wherein the detection of the labeled DNA or RNA is indicative of the presence of a nucleic acid molecule encoding a mutated FGFR-4 in the sample.

18. The method of claim 15, which method comprises contacting the sample of nucleic acid with a restriction enzyme whose recognition sequence is affected by the mutation in the mutated FGFR-4 and detecting the presence or absence of fragments or the presence of altered fragments of the nucleic acid after contact with the restriction enzyme, wherein the absence of fragments or the presence of altered fragments of the nucleic acid after contact with the restriction enzyme is indicative of the presence of a nucleic acid molecule encoding a mutated FGFR-4 in the sample.

19. The method of claim 18, wherein the mutation in the mutated FGFR-4 occurs at amino acid position 388.

20. A method of identifying an inhibitor of a mutant FGFR-4, which method comprises contacting a potential inhibitor with a mutated FGFR-4 and determining activity in

the absence and presence of the potential inhibitor, wherein a decrease in activity in the presence of the potential inhibitor indicates that the potential inhibitor is an inhibitor of mutant FGFR-4.

21. The method of claim 20, wherein the activity is tyrosine kinase activity.

22. The method of claim 20, wherein the activity is interaction with a protein with which FGFR-4 interacts.

23. A method for the prophylactic and/or therapeutic treatment of a RTK-hyperfunction-induced disorder in a mammal, which method comprises administering to a mammal in need thereof an effective amount of at least one inhibitor of a mutant FGFR-4 identified in accordance with the method of claim 20, wherein said RTK-hyperfunction-induced disorder is treated prophylactically and/or therapeutically.

24. A method for the prophylactic and/or therapeutic treatment of a RTK-hyperfunction-induced disorder in a mammal, which method comprises administering to a mammal in need thereof an effective amount of at least one inhibitor of a mutant FGFR-4 identified in accordance with the method of claim 21, wherein said RTK-hyperfunction-induced disorder is treated prophylactically and/or therapeutically.

25. A method for the prophylactic and/or therapeutic treatment of a RTK-hyperfunction-induced disorder in a mammal, which method comprises administering to a mammal in need thereof an effective amount of at least one inhibitor of a mutant FGFR-4 identified in accordance with the method of claim 22, wherein said RTK-hyperfunction-induced disorder is treated prophylactically and/or therapeutically.

26. An antibody that reacts specifically with a mutated FGFR-4 receptor, which is overexpressed and/or has altered activity in a cell.

27. The antibody of claim 26, wherein a hydrophobic amino acid in the wild-type receptor has been exchanged for a hydrophobic amino acid in the mutated FGFR-4 receptor.

28. The antibody of claim 26, wherein the transmembrane domain of the mutated FGFR-4 receptor comprises a point mutation.

29. The antibody of claim 28, wherein the point mutation occurs at amino acid 388 and, optionally, results in replacement of glycine with arginine.